Editorial Commentary

The inflammatory potential of diet and bladder cancer risk: results from a prospective cohort study

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With an estimated 430,000 new diagnoses and 165,000 deaths per year worldwide (1) and per patient treatment costs of over 100,000 US dollars (2), bladder cancer represents a major burden for patients and society. From a public health perspective, it would be desirable to identify preventable risk factors. One known preventable risk factor for bladder cancer is smoking (1), causing inflammation which is known to play an important role in the biology of many tumors and is considered a hallmark of cancer (3,4). In bladder cancer, inflammation plays a role in prognosis (5) and therapy [e.g., intravesical Bacillus Calmette-Guérin (6) or checkpoint inhibitors (7)].

Inflammation is also influenced by nutrition, which is a known risk factor for several malignancies. However, studies regarding associations between nutrition and bladder cancer report conflicting results (8). Abufaraj *et al*. report a statistically significant association between a nutrition score related to inflammation and bladder cancer risk in the crude analysis (9). However, this association was attenuated and became non- statistically significant after controlling for known confounders.

The specific nutrition score examined was the Empirical Dietary Inflammatory Pattern (EDIP). This score was developed using reduced rank regression, which is a statistical method that determined linear functions of food groups by maximizing the explained variation in the responses of plasma inflammatory markers (10). Validation of the EDIP in an independent cohort showed only a weak (r=0.09–0.14) association with plasma inflammatory markers (11). Consequently, this score might serve as a proxy for inflammation only to a very limited extent and should therefore be regarded as subject to non-differential misclassification.

However, the absence of evidence of this study may not serve as evidence for absence of a true effect (12): this result could either be based because there is no association or because the study still had insufficient power (sample size and/or follow-up) to demonstrate a significant measure of association. Because carcinogenesis is a slow process, any exposure needs decades to have an impact and because the estimated effect in nutritional/environmental health science tend to be small, the power needs to be huge. This latent effect and expected small effect sizes is a potential explanation for discrepant findings from other observational studies and randomized trials from the same institution (13,14). In conclusion, the authors should be congratulated for pulling together three data set including over 200,000 patients and nearly 5 million person-years of observation and for their continuing efforts to maintain unique cohorts and develop novel metrics of dietary patterns. We believe that nutrition is a promising field and hope that one day recommendations reduce the impact of bladder cancer on individuals and society.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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